

Running Head: The Effect of Maternal Depression on Birth Outcome and Child Obesity
Risk

A proposal to the National Institute of Health

by

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Abstract

Objective: There is a large body of research on the cognitive, emotional, and behavioral outcomes of children of depressed mothers (Ramchandani, 2011). However, little research has been done on whether maternal perinatal depression affects child obesity. Thus, the proposed study will be critically relevant to the current public health epidemic of obesity. The objective of the proposed study will be to examine the correlation between maternal depression and child obesity risk.

Setting: 3,000 mother-child pairs in Project VIVA, a prospective cohort study, located in Boston, Massachusetts.

Study Design: At the first clinical visit, mothers will take a survey of the risk factors for depression and basic demographic information will also be collected. At the mid-pregnancy visit, the Edinburgh Postnatal Depression Scale (EPDS) and Binge Eating Scale will be administered. All previous variables will be updated again 3-months postpartum, 6-months postpartum, and annually for 3 years. At birth, the Neonatal Behavioral Assessment Scale (NBAS) will be used to generally evaluate the health of the newborn. In lieu of the standard BMI, we will use a more precise and comprehensive tool, Pea Pod, to measure body fat composition of the newborn. Information about the child's social environment and lifestyle will be collected via questionnaires and interviews annually until age 3. The study will be conducted in compliance with the 1990 Institute of Medicine guidelines. Human subjects committee of Harvard Pilgrim Health Care will also approve the study protocols.

Predicted Results: The groups will be separated as 'depressed' versus 'not depressed.' ANOVA will be used to analyze the results. Current literature supports

our hypothesis: mothers with perinatal depression will have greater risk factors at pre-pregnancy, higher Edinburgh Post Partum Depression scale score (depression as 12+), and higher Binge Eating score (severe bingeing 27+). We also predict that infants exposed to perinatal depression will weigh less at birth, have lower NBAS scores at birth and higher percent body fat at age 3.

Conclusion: The study will contribute to the existing knowledge of perinatal risk factors that contribute to early onset obesity. New recommendations may also be made to prevent maternal perinatal depression.

Resources: The study will be a collaborative effort with Project Viva longitudinal study under the supervision and guidance of Matthew W. Gillman, MD, of Harvard Medical School and his research team at Harvard Pilgrim Health Care.

Key Words: perinatal depression, pregnancy, obesity, birth outcome, risk factors, Edinburgh Depression Scale, Binge Eating scale, Pea Pod, Neonatal Behavioral Assessment Scale

Research Aims

The recent prevalence of obesity has become a pressing public health concern. As reviewed in Selassie & Sinha (2011), data from the National Health and Nutrition Examination Survey (NHANES) conducted in 2007 – 2008 reported that 68% of U.S. adults aged 20 – 74 years old are either overweight or obese. It was recently found that early life exposures might be a risk factor for obesity in adulthood. More specifically, obesity during adolescence increases the likelihood of being overweight in later life (The, 2010). Additionally, obese individuals are at greater risk for developing more health problems, such as type 2 diabetes, coronary artery disease, hypertension, asthma, orthopaedic problems, sleep apnea and psychosocial difficulties (cited in Ertel, 2010). People with obesity also report lower quality of life (Puhl & Brownwell, 2011). It is for this reason understanding the complex origins of obesity is a critically important area of research.

“At similar alarmingly high rates is the prevalence of major depressive disorder (MDD), one of the most common psychiatric illnesses in the adult population” and also a risk factor for obesity (Cizza, 2011). Depression is common not only in the average adult, but in expecting mothers as well. There exists a 10 – 15% prevalence of perinatal depression in the general population (Meltzer-Brody, 2011) and, yet, little research is conducted on the topic (Goodman, 2010). We already know that untreated perinatal depression can lead to birth complications such as low-birth weight deliveries (Rich- Edwards, 2006), but little is known on the effect that untreated depression has on obesity risk. Thus, greater knowledge about the outcomes of early exposure to depression is particularly important.

Furthermore, there is compelling evidence to believe there is an undiscovered relationship between obesity and depression. In particular, one study found that depressed individuals have a fat mass obesity gene, FTO, located on chromosome 16 (Rivera et al, 2011). This suggests there is a genetic contribution to obesity. Despite the fact that perinatal depression is the leading cause of complication in childbirth (Gaynes, 2005), research on detection and treatment of depression is still considerably lacking (Goodman, 2010). The dearth of information in this particular domain of research is a frightening reality.

Put together, the existing knowledge on depressed and obese people suggests that there is an important association that needs to be discovered. Despite many public health efforts to understand the risk factors for childhood obesity, few studies have examined the effect that early exposure to perinatal depression has on birth outcome and childhood obesity risk. Consequently, there is a dire need to address this gap in the scientific knowledge. The proposed study aims to fill that void.

The proposed study, in general, aims to contribute to the public health effort to understand the risk factors that result in early onset of obesity. Specifically, though, the objective is to test the hypothesis that the fetus with early exposure to perinatal depression will have a greater risk for childhood obesity. The study will contribute to the existing body of knowledge in a novel way. Not only will the prospective study perform statistical analysis in an original way, but it will also introduce an innovative method to measure body mass. In this way, the study will make a significant contribution to the literature.

Literature Review & Significance

Not only has the prevalence of obesity reached epidemic proportions, but also child obesity, in particular, is considered a major threat to children's health in the US (cited in Schwartz, 2003). It was found that 11% of children ages 6 – 17 years are obese as estimated by the BMI > 95th percentile (cited in Schwartz, 2003). Studies have also shown that there is a persistence of obesity from childhood to adulthood (Micic, 2001). Despite the startling statistics on obesity, there is still a strong need for the early detection of factors contributing to development and maintenance of obesity (Puder, 2010). Research has also shown that obese children are more likely to be depressed (cited in Puder, 2010) and suffer from chronic diseases such as diabetes, hypertension, and cardiovascular disease (Huang, 2011). While there is an expanding body of knowledge on the adverse effects of obesity, there is limited knowledge on the early exposures that contribute to obesity. It was believed that treatment and prevention were the primary responsibility of the child and their parents (Schwartz, 2003); however, recent research suggests that environmental determinants interact with genetic, behavioral, and biological factors to induce weight gain in individuals (Melendez, 2011). It is for this reason understanding the risk factors that contribute to child obesity is a pressing research question, one effectively addressed by the proposed study.

Growing evidence also suggests that obesity and diabetes is elevated in people with depression (cited in Rivera, 2011). A study by Farmer et al. (2008) found a relationship between clinical depression, body mass index (BMI), type 2 diabetes, coronary heart disease and hypertension (cited in Rivera, 2011). The fat mass and

obesity – associated gene, FTO, on chromosome 16q has been found several times to contribute to human obesity (cited in Rivera, 2011). Rivera's (2011) work was the first study to investigate FTO and BMI within the context of MDD, and results indicated that a history of depression moderates the effect of FTO on BMI. This finding strengthens the association of depression and obesity, as it suggests that that there is a biological mechanism underlying the mood and weight gain. However, the study has a few limitations. One of the clinical samples used in the study recruited 974 men and only 724 women. This unequal gender distribution does not help us draw any conclusions about how chronic depression in women moderates the effect of FTO on the BMI. A larger sample size, or at least an equal ratio of men- women in the study, would have been constructive. Secondly, BMI for one of the groups was calculated from a self- report of weight and height that was collected over the phone. While this was a compelling finding in its own right, we must critically question the internal validity of this study. In addition, the researchers used the Beck Depression Inventory for participation. External validity is also in question, since a clinical definition of depression was not used and a survey was used as primary means of information. The study also did not mention if women were intending to get pregnant during their participation, so little is known about the pregnancy status of women recruited for this study.

Rivera (2011) was not the only one who found a relationship between depression and obesity. In fact, Cizza (2011) also found that major depressive disorder (MDD) – a common psychiatric illness in the adult population – is a risk factor for cardiovascular disease, low bone mass, and obesity in premenopausal

women. This large prospective study evaluated if depression as a premenopausal women is a risk factor for obesity-related diseases. This study sheds more light than Rivera's work on depression and obesity in women. However, the subject pool is limited to premenopausal women and does not account for antenatal or postpartum depression. In addition, the researchers also measured obesity by BMI, calculating the typical weight to height ratio. The study measured many negative effects of Major Depressive Disorder, in which obesity was simply one of many of those effects. That is to say, this study did not focus on depression as it relates to obesity. Since women in the study were not pregnant, these effects cannot be attributed to the fetus in any way. This is all to say that despite compelling findings, little is still known about the effects of early exposure to maternal depression and this remains an unfulfilled gap in the literature.

Several studies on psychosocial stressors also hint at an undiscovered relationship between depression and obesity. Perinatal depression is just as common as depression in the adult population. Specifically, Meltzer-Brody (2011) found that perinatal depression exists in 10 – 15% of the general population. This study examined the pathogenesis and treatment of depression during pregnancy (Meltzer-Brody, 2011). It did not, however, evaluate ways to prevent maternal depression or notice any effect of maternal depression on the developing fetus or the child. In fact, the researchers themselves admit that the potential long – term impact of perinatal depression on the developing fetus is still unknown (Meltzer-Brody, 2011). Given the increasing prevalence of maternal depression, this gap in the literature is unsettling.

Additional studies found a correlation between psychosocial stressors during pregnancy and birth outcome (cited in Hobel, 2008). Studies show that that psychosocial stressors (both acute and chronic) lead to the two major adverse pregnancy outcomes: preterm births and low birth weight (cited in Hobel, 2008). This study did not examine depression specifically and studied stressors in general. While the researchers measured birth outcome, they did not find any longitudinal effect of psychosocial stressors on the developing fetus. While the study emphasized that stressors contribute to birth outcome, results actually showed it was the mother's perception of the stressor that contributed to the birth outcome.

In particular, the study (cited in Hobel, 2008) showed that life stressors affect birth outcomes when they occur in the first trimester. As an example, Hobel (2008) cited Glynn et al's (2004) work that pregnant women exposed to the Northridge earthquake in California during the first trimester of the pregnancy had a shorter gestational age at delivery. While the studies make significant contribution of stressors on birth outcome, Glynn et al (2004) did not examine depression in particular as it related to birth outcome. In addition, the study was not a longitudinal overview of the long-term effects of stressors in that the study just measured gestational age. Moreover, the study did not look at stressors unrelated to a traumatic event; the studies evaluated birth outcome of mothers who experienced the Northridge earthquake, the World Trade Center on September 11, 2001, and Ukrainian Chernobyl disaster just to name a few (cited in Hobel, 2003). There is a wide-range of stressful experiences and perceptions of those experiences also vary. Given the small sample size of 307 women in the study, the question of external

validity is raised. The participants receiving medical care for depression were also excluded, so women on anti-depressants were not studied. Little can be said about birth outcomes of stressors faced by women on anti-depressants.

Hobel is just one of many researchers that did not focus on depression and birth outcome. According Figueiredo (2011), there may be a reason for this. Studies cited in Figueiredo (2011) show that more than 25% of pregnant women have high-anxiety compared to just over 15% of pregnant women with depression. It can be assumed that since more expecting mothers suffer from anxiety, rather than depression, few researchers have taken the time to investigate the effects of maternal depression.

The studies that have examined the role of anti-depressants did not measure how SSRI's affect child weight gain or child obesity. One study found that length of SSRI exposure in utero affects mental and psychomotor development of child at age 3 (Casper, 2011). While this study followed the child until age 3, the researchers did not examine if SSRI exposure contributes to child obesity in any way. Casper (2011) also performed a quick assessment of the newborn using the Apgar score, a measurement used mainly to evaluate if newborn requires additional care (Apgar, 1953). It is not the most efficient means for research purposes. Given this finding, we have a better understanding of the effect that SSRI's have on child development. However, there is still uncertainty about the long-term effects of SSRI's and the effect that SSRI's have on areas of development other than cognitive development.

Although presently undiscovered, the literature makes clear that there may be a relationship between depression and obesity. Given the particular gaps in the

literature – few have acknowledged the effect of perinatal depression on child obesity – the proposed study will surely make a unique contribution to the body of knowledge. The study aims to test the hypothesis that the fetus with early exposure to perinatal depression will have a greater risk for child obesity. The specific contributions are detailed below:

1. Perinatal depression

- a. The proposed study, unlike prior studies, will follow a participant from pre-pregnancy until post-partum. The studies that examined depression during pregnancy also included anxiety (Glynn et al, 2004; Figueiroado, 2007). Still other studies noticed the link between depression and other health problems as they relate only to the woman, not the fetus or child (Cizza, 2011). The proposed study will account for these losses and explore how an early exposure to perinatal depression may affect the developing fetus and child in later-life – a research area previously unexamined.

2. Novel way to measure body mass

- a. Most studies have used Body Mass Index (BMI) to define obesity (Burdette, 2003; Chen, 2010; Ertel, 2010). The simple proportion of height and weight, however, is not sufficient to get an accurate and precise measure of body mass. Instead, the proposed study will utilize the latest technology called “Pea Pod” and “Bod Pod” that uses air displacement and accounts for muscle mass, fat mass, tissue mass, and

bone density for the most accurate measure of body composition (Ellis, 2007).

3. Non-traditional method to analyze the results

- a. In past studies, many researchers have used a multivariable regression models to find an association with depression and child BMI score (Rivera, 2011; Ertel, 2010; Rich-Edwards, 2006). On the other hand, the proposed study will use ANOVA to evaluate the correlation that depressed versus non- depressed mothers have on child obesity risk. This simple ANOVA comparison will help to understand if a correlation exists without analyzing any additional factors.

Method and Design

Subject Recruitment

Participants for the proposed study will be recruited from Project Viva, a prospective cohort study of pregnancy outcomes and maternal and child health (Rich-Edwards, 2006). Prospective mothers will be recruited during a pre-pregnancy visit at 1 the 8 offices of the Harvard Vanguard Medical center, a large multi-specialty group practice and comprehensive wellness center located in Boston. Data will be collected at 3 times during the pregnancy: at the first clinical visit, at the mid-pregnancy visit, 3 – months postpartum, 6 – months postpartum, and then annually for 3 years. All data will be collected during the participant's routine check- up at the medical office or at home for the participant's convenience. No situations or materials will be considered invasive at any time during the study. The participants will experience little, if any, risk during the study.

Research Design

Research assistants will recruit 3,000 subjects attending their initial visit at the HVMO health care system. 1,000 subjects will be assigned to 1 of 3 possible groups: clinically depressed (taking antidepressants), depressive symptoms, and a control group of subjects with no known mental health or physical complaints. Medical records will be checked to verify depression and a clinical psychologist will measure depression (or lack thereof) in all participants against DSM-IV criteria before officially enrolling subjects in the study.

Exclusion Criteria

VIVA will employ their own exclusion criteria for the participants. Eligibility criteria for the VIVA project includes fluency in English, gestational age <22 weeks, and singleton pregnancy (Oken, 2007). Participants will also be excluded if any of the following apply: a) participant smokes or uses alcohol/drugs; b) depression is not the primary mental health problem; c) take vitamin supplements to relieve depressive symptoms; d) delivery of preterm births.

Project Timeline

During the initial visit, we will obtain consent from the participants. Consent will be obtained again at each visit to ensure continued interest and participation in the study. We will review a research consent checklist with the participants (Appendix A). Participants will also complete a survey for risk factors of depression (Appendix B). The survey will ask a series of questions (as per findings of risk factors for depression by Rich- Edwards, 2005) about the participant's education level, household income, record of financial hardship, immigration status, and race/ethnicity. Any participant with missing data on the surveys or questionnaires will be excluded from the study. The importance of complete and honest answers to all surveys will be relayed to participants upon enrollment.

At the mid-pregnancy visit, participants will complete the 10 – item Edinburgh Postpartum Depression scale (Cox, 1987) as in Appendix C. A 16 – item Binge Eating Scale will also be part of this second visit. Variables from the first visit will be updated during again and any changes in the living environment will be noted. A brief interview with the participant will be used to collect information

about such as daily physical activity, vitamin use, and eating behaviors including new cravings or allergies since pregnancy.

During pregnancy, we will collect basic medical information about the new born (height, weight, etc) from the medical chart. In addition, we will collect NBAS and Pea Pod measurements for a behavioral assessment at birth and a measure of the body fat distribution, respectively.

At the 3- months post- partum visit, 6 – months post- partum visit, and annually for 3 years, the interviews will be conducted and variables from previous visits will be updated to ensure internal validity.

An ANOVA will be used to assess correlation between each mother- child pair, where mothers will be categorized as ‘depressed’ or ‘ not depressed.’ All things considered, the study will take approximately 4 years to complete starting in 2012.

I. Rationale for Design & Methods

a. Longitudinal Study

- i. Despite the fact that the proposed study is low time commitment with no known risk involved, the proposed study will only follow a child for no more than 3 years. Typically, Project VIVA studies collect data from a child until age 10. We believe we will have adequate information by age 3; extending the study to age 10 would be unnecessary for our purposes. The precision of body fat distribution using the Pea Pod measurement and consistent

interviews and follow- up questionnaires will give us sufficient information.

b. Project VIVA collaboration

- i. There is strong reason to collaborate with Project VIVA, a well-funded longitudinal study on mother and children. Our hypothesis compliments the VIVA team's motivation to understand how maternal lifestyle choices, such as diet and certain environmental factors, contribute to birth outcome and child health. In addition, the collaboration will be cost-effective, since participants are readily accessible and little budget money will be spent on advertising for study recruitment. We will certainly expend our efforts to enroll a more diverse demographic group than presently associated with Project VIVA to ensure external validity. According to a study by Davis (2011), African Americans and low socioeconomic populations are at greater risk for depression and obesity, so our study will be sure to represent this population.

c. Exclusion Criteria

- i. In addition to the criteria upheld by the Project VIVA study, our study will also exclude woman who smoke, drink, or use drugs. Since we know that women who smoke and drink alcohol have poor pregnancy outcome (Cogswell,

2003), we will exclude them to focus our study on the effect of maternal depression without the various self-mediating influences (other than Binge Eating, of course). Project VIVA already excludes participants with deliveries at < 22 gestational weeks and, similarly, we will be sure to exclude mothers with preterm births and babies with congenital anomalies as they will require intensive and complicated care after birth (Kelly, 2006). Project VIVA exclusion criteria already includes singleton pregnancy and we will also be sure that mothers with twins will be excluded from the study. We will also exclude woman whose primary mental health problem is not depression. The focus of the proposed study is on maternal depression. Even if obese mothers have depression, they will be excluded because it is best not to have competing risk factors. Maternal obesity is in itself a risk factor (Chen, 2010). Since the study focuses on the depression and obesity association, excluding confounding variables, such as obesity, would be best in this particular study.

d. Specific Measurement Tools

i. Combination of DSM-V interview & EPDS for depression

1. In addition to verifying history of depression on the medical charts of the participants in the depressed

group, we will also conduct a clinical interview using DSM-V criteria. A psychologist or psychiatrist from Harvard Vanguard Medical group will conduct the interview at the first visit before enrollment. EPDS is simply a screening tool for depression, yet many studies have used it as the primary means to measure depression (Ertel, 2010). Thus, the DSM – V interview for clinical depression makes our study design especially comprehensive.

ii. Risk factor for Depression questionnaire

1. A Project VIVA study conducted by Rich- Edwards (2005) found that the two risk factors for perinatal and postpartum depression are financial hardship and unwanted pregnancy. It is for this reason the questionnaire (Appendix A) asks about household income and intent of pregnancy. Risk factors will be reviewed after the study to follow any trends in risk factors for maternal depression and child obesity.

iii. Edinburgh Postnatal Depression Scale

1. This particular scale will be used because it has been approved for antenatal and postpartum depression (Ertel, 2010). EPDS is preferred over the Beck Depression Inventory because of the concentrated

effort to understand perinatal depression. Beck Inventory is useful to study severity of depression or if participants are both male and female.

iv. Binge Eating Scale

1. Since psychological stress is shown to play a role in binge-eating behavior (Colles, 2008), it is important to understand binge-eating behaviors of depressed mothers. This finding by Colles (2008) leads us to predict that binge eating will be used to self-medicate in depressed mothers.

v. Neonatal Behavioral Assessment Score (NBAS)

1. The NBAS is a comprehensive behavioral assessment of birth outcome. NBAS predicts mental and psychomotor development (Sans et al, 2011). Since it has been found that exposure to SSRI's during pregnancy effects mental and psychomotor development of the child (Casper, 2011), this is an appropriate instrument for the study. NBAS will be used in lieu of the Apgar score. Since the Apgar score is designed for a single observer to quickly evaluate the general condition of the child for proper care and treatment (Apgar, 1953), it is

effective measurement for care management not necessarily for documentation in research studies.

vi. Pea Pod & Bod Pod Measurement

1. Most all research studies in this area have evaluated BMI or skin-fold measurements (Burdette, 2003; Davis, 2011; Topham, 2009). The proposed study will introduce a more innovative and accurate way to measure body fat composition, using fat mass, lean tissue, muscle mass, and bone weight to measure nutrition in not more than 5 minutes (Ellis, 2007). It is effective for newborns and adults alike.

Conclusion

Predicted Results

SES demographics show that financial hardship and unwanted pregnancy are risk factors for antenatal and postpartum depression (Rich-Edwards, 2006). Studies also show that antenatal and postpartum depressions are associated with early child adiposity (Ertel, 2010). In addition, we already know that permissive parenting styles of depressed and obese mothers leads to child weight gain (Burdette, 2003). Given these findings, we predict that depressed mothers will have greater number of risk factors as assessed by the pre-pregnancy interview, higher Edinburgh Depression scale score (12+ denotes highly depressed), and higher Binge Eating Score (27+ denotes severe binging).

Prior research by Chen et al. (2010) tells us that maternal obesity influences the Apgar score at birth. In particular, obese mothers are more likely to have obese children. Our predicted results will compliment this finding. We predict that infants of depressed mothers will have: smaller weight at birth, lower NBAS scores at birth and higher % body fat on Bod Pod measurement as newborn and Bod Pod measurement at age 3. Consistent with a similar Project VIVA study by Ertel (2010), our hope is that the results will raise the possibility that treating perinatal depression will reduce obesity in childhood.

Limitations

There are a few limitations that we predict with this study. Firstly, the demographics of the participants in the Project VIVA study are limited. In particular, 92% mothers are Boston residents; average age is 32 years; and merely 22% were born outside the US and majority of the participants associate with a high socioeconomic class (Ertel, 2010). One of the reasons we will collaborate with Project VIVA was for the convenience and accessibility of recruiting subjects. In order to avoid this as much as possible, we do have our own specific criteria for recruitment. In particular, we will recruit women from a wide-ranging socioeconomic demographic and maternal education level for external validity (Davis, 2011). Since most of the information is collected by questionnaires, surveys, or in-person clinical visits at the Harvard medical offices, it is inconvenient to both parties to enroll subjects from outside the Boston area. We would be limited to phone interviews and percent body fat of newborn and infant would be difficult to

calculate. Against this, we will make considerable effort to recruit a diverse group in Boston.

Separating genes and environment is always a difficult task and this will pose a difficulty in our proposed study as well. We have thought about this considerably in designing the study and to evade the issue, we decided to set rigorous exclusion criteria. Our hope is that this will limit the confounding factors. Even with a considerably large population size of 1,000 participants in each group, the sample size is still too small to make any definitive or causal claims. Future work will collaborate with the National Child's Study to recruit a larger sample size.

Another limitation may be of one internal validity, specifically, our data collection method. The questionnaires, surveys, and brief interviews let us evaluate depression at different stages (i.e., first clinical visit, mid-pregnancy, 3 months post partum and 6 months post partum etc). This enables us to predict which stage in the pregnancy the depression is more prevalent and which stage is the 'critical period' for obesity risk. Still, however, the data we collect requires cautious evaluation because it depends on self-report. Against this, we will crosscheck information with the medical chart. Still, however, it is still possible that information is under or overestimated. It is for this reason we will exclude women if any part of the surveys or questionnaires are not filled out and/or appear inconsistent. Although direct observation of lifestyle and basic SES demographics would be a more valid measure, the study design was developed for convenience to the participants. Participants will not need to spend more time contributing to the study other than attending

regularly scheduled physician's visits. It is for this reason we will update variables at each visit to ensure reliable information.

Future Studies

There are an infinite number of avenues for future research that may address the problem of childhood obesity. For the interest of this paper topic, though, there are just a few that we would like to address. Since obesity is a pressing concern in public health today, conducting only correlation studies may be unnecessary. Instead, it is critically important to test the efficacy of different interventions and treatments for maternal depression and child obesity. We know that psychosocial stress negatively affects pregnancy outcome (Hobel, 2008). One option is to educate upcoming physicians on the negative effects of maternal depression on mother and child health and encourage them to address psychological health with expecting mothers at each clinical visit. Future studies may also test the efficacy of psychological evaluation as standard of care for expecting mothers. Moreover, future studies may test the efficacy of an education program for children – educating them in school or after-school care programs about and obesity risk – and a mandatory school project on obesity prevention. The hope is that focused education in the school system will encourage children to healthy and nutrition eating behavior and lifestyle practices to sustain good health into adulthood.

Conflicts of Interest

No known conflicts of interest will interfere with this study.

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References

- Apgar Virginia. (1953). A proposal for a new method of evaluation of the new born infant. *Current Research in Anesthetic Analgesia*, 32, 260-267.
- Burdette Hillary L., Whitaker Robert C., Kahn Robert S., Harvey – Berino Jean. (2003). Association of Maternal Obesity and Depressive Symptoms with Television- Viewing Time in Low- Income Preschool Children. *Pediatrics & Adolescent Medicine*, 157, 894-899.
- Casper Regina. (2011). Length of prenatal exposure to selective serotonin reuptake inhibitor (SSRI) antidepressants: effects on neonatal adaptation and psychomotor development. *Psychopharmacology* [Epub ahead of print]
- Chen Minghua, McNiff Ceare, Madan Juliette, Goodman Elizabeth, Davis Jonathan M., Dammann Olaf. (2010). Maternal obesity and neonatal Apgar scores. *The Journal of Maternal- Fetal and Neonatal Medicine*, 23, 89 – 95.
- Cizza G. (2011). Major Depressive Disorder is a risk factor for low bone mass, central obesity, and other medical conditions. *Dialogues Clinical Neuroscience*, 13, 73 – 87.
- Cogswell Mary E. (2003). Cigarette Smoking, Alcohol Use and Adverse Pregnancy Outcomes: Implications for Micronutrient Supplementation. *American Society for Nutritional Sciences*, 133, 1722 – 1731.

Colles SL, Dixon JB, O'Brien PE. (2008). Loss of control is central to psychological disturbance associated with binge eating disorder. *Obesity (Silver Spring)*, 16, 608 – 614.

Cox, J.L., Holden, J.M., and Sagovsky, R. (1987). Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry*, 150, 782-786.

Davis Melvin, Youg LaShun, Davis Sheila P., Moll George. (2011). Parental Depression, Family Functioning, and Obesity among African American Children. *The Association of Black Nursing Faculty Journal*, 4 – 5.

Ellis, Kenneth, Yao Manjiang, Shypailo Roman J., Urlando Alessandro, Wong William W, Heird William C. (2007). Body – composition assessment in infancy: air – displacement plethymosgraphy compared with a reference 4 – compartment model. *American Journal of Clinical Nutrition*, 85, 90 -95.

Ertel Karen A., Koenen Karestan C., Rich-Edwards Janet W., Gillman, Matthew W. (2010). Antenatal and postpartum depressive symptoms are differentially associated with early childhood weight and adiposity. *Paediatric and Perinatal Epidemiology*, 24, 179 – 189.

Figueiredo Barbara & Conde Ana. (2011). Anxiety and Depression in women and men from early pregnancy to 3- months post partum. *Women's Mental Health*. [Epub Ahead of Print].

Gaynes BN, Gavin NI, Meltzer-Brody S, et al. (2005). Perinatal de-pression: Prevalence, screening accuracy, and screening outcomes. *Evidence Report Technology Assessment (Summary)*, 119, 1–8.

Goodman Janice H & Tyer – Viola Lynda. (2010). Detection, Treatment, and Referral of Perinatal Depression and Anxiety by Obstetrical Providers. *Journal of Women's Health*, 19, 477 – 480.

Hobel Calvin, Goldstein Amy, & Barrett Emily. (2008). Psychosocial Stress and Pregnancy Outcome. *Clinical Obstetrics and Gynecology*, 51, 333 – 348.

Huang CY, Chi SC, Sousa VD, Wang CP, & Pan KC. (2011). Depression, coronary artery disease, type 2 diabetes, metabolic syndrome and quality of life in Taiwanese adult in a cardiovascular department of a major hospital in Southern Taiwan. *Journal of Clinical Nursing*, 20, 1293 – 12302.

Kelly Michelle. (2006). The Basics of Prematurity. *Journal of Pediatric Health Care*, 4, 238 – 244.

Melendez Guillermo. (2011). Introduction: The First Forum on Child Obesity Interventions. *American Society for Nutrition*, 2, 157 – 158.

Micic D. (2001). Obesity in children and adolescents – a new epidemic? Consequences in adult life. *Journal of Pediatric Endocrinology*, 14, 1345 – 1352.

Meltzer- Brody S. (2011). New Insights into perinatal depression: pathogenesis and treatment during pregnancy and postpartum. *Dialogues in Clinical Neuroscience*, 13, 89 - 100.

Oken Emily, Taveras Elsie M., Kleinman Ken P., Rich-Edwards Janet W., & Gillman Matthew W. (2007). Gestational weight gain and child adiposity at age 3 years. *American Journal Obstetric Gynecology*, 4, 1-8.

Oken Emily, Huh, SY, Taveras EM, Rich-Edwards JW, & Gillman MW. (2005). Associations of maternal prenatal smoking with child adiposity and blood pressure. *Obesity Research*, 11, 2000 - 2008.

Puder JJ & Munsch S. (2010). Psychological correlates of childhood obesity. *International Journal of Obesity*, 34, 37 – 43.

Puhl R & Brownell K. (2010). Bias, discrimination, and obesity. *Obesity Research*, 9, 788–805.

Ramachandani Paul G, Lamprini Psychogiou, Vlachos Haido, Iles Jane, Sethna Vahestha, Netsi Elena, Lodder Annemarie. (2011). Paternal Depression: An Examination of Its Links With Father, Child and Family Functioning in the Postnatal Period. *Depression and Anxiety*, 1 – 7.

Rich-Edwards, Janet W et al. (2006). Sociodemographic predictors of antenatal and postpartum depressive symptoms among women in a medical group practice. *Journal of Epidemiological Community Health*, 60, 221 – 227.

Rivera M, Cohen – Woods S, Kapur K, Breen G, Ng My, Butler AW, Craddock N, Gill N, Korszun A, Maier W, Mors O, Owen MJ, Preisig M, Bergmann S, Tozzi F, Rice J, Rietschel M, Rucker J, Schosser A, Aitchison KJ, Uher R, Craig IW, Lewis CM, Farmer AE, McGuffin P. (2011). Depressive disorder moderates the effect of the FTO gene on body mass index. *Molecular Psychiatry*, 11, 1- 8.

Sans JC, Hernandez-Martinez C, Esparó G, Fernandez- Ballart J. (2011). Neonatal Behavioral Assessment Scale as a Predictor of Cognitive Development and IQ in full-term infants: A 6- year longitudinal study. *Acta Paediatrica*. [Epub ahead of print]

Selassie Meron & Sinha Ashish. (2011). The epidemiology and aetiology of obesity: a global challenge. *Best Practice & Research Clinical Anesthesiology*, 25, 1– 9.

Schwartz M.B. & Puhl R. (2003). Childhood obesity: a societal problem to solve. *Obesity Reviews*, 4, 57 – 71.

The Natalie S., Suchindran C, North KE, Popkin KE, Gordon – Larsen P. (2010). Association of adolescent obesity with risk of severe obesity in adulthood. *Journal of American Medical Association*, 18, 2042 – 2047.

Topham Glade L., Page Melanie C., Hubbs-Tait Laura, Rutledge Julie M., Kennedy Tay S., Shriver Lenka, Harrist Amanda W. (2009). Maternal depression and socio-economic status moderate the parenting style/child obesity association. *Public Health Nutrition*, 8, 1237 – 1244.